# ATOC 3500/CHEM 3151 - Week 10
# Urban Pollution

<u>Back to the troposphere (Chapters 3, 8 and 11)</u>

(Where to go for the basics – most of this we've already done)

- Introduction (p 206-208)
- Sources, Sinks, Transport (p 25-28, p 29-33)
- Dry and Wet Deposition (p 33-35)
- The Boundary Layer (p 100)
- Transport in the Troposphere (p 37-40)
- Oxidation (p 99-104)
- Ozone Production and NOx (p 104-105)

This lecture – why we care

# Health and welfare effects of air pollution (p 206-211)

- Pollution episodes, Cause and effect relationships
- Human body
- Health effects of regulated pollutants
- Personal air pollution (smoking)
- Risk assessment and management
- Effects on agricultural crops, ornamental plants, and trees
- Effects on domesticated animals, Ecological effects
- Effects on materials
- Odor pollution

# What are air pollution “episodes?”

Characterized by significant short-term increases in atmospheric pollutant concentrations above normal daily levels

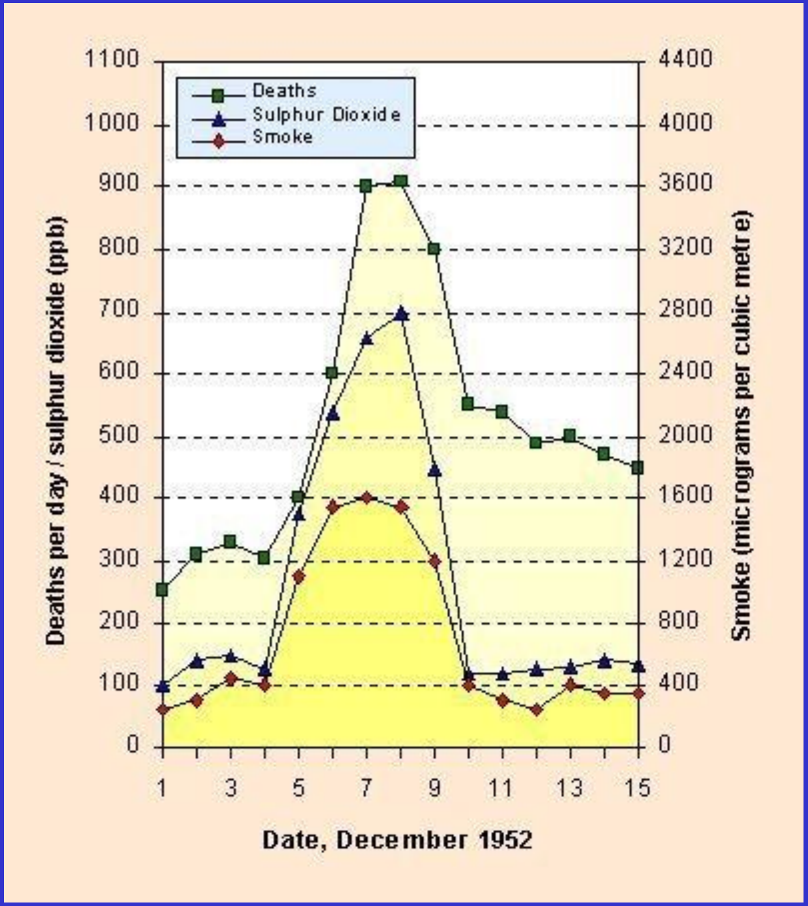
Vary in intensity from mild (limited public health concern) to severe (widespread illness and mortality)

1952 – London, 4000 excess deaths attributed to pollution.

Many other examples that are less dramatic, but that provide solid evidence that elevated pollutant levels can cause illness and death

A problem for both developed and developing countries

London, December 1952
(see page 210-211)




Primarily due to sulfur, tars (heavy hydrocarbons), and hydrocarbons from bituminous coal

# Epidemiological Studies (p 141-149)

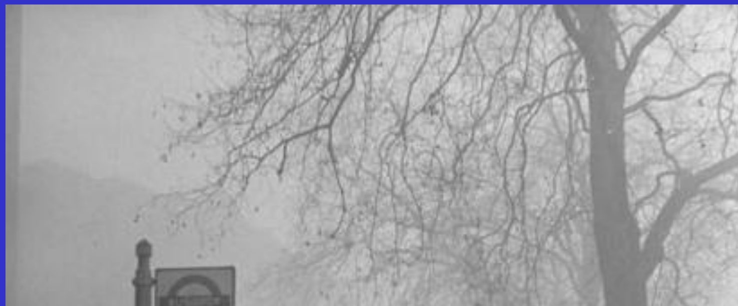
Statistical relationship between disease in a population and potential causal factors

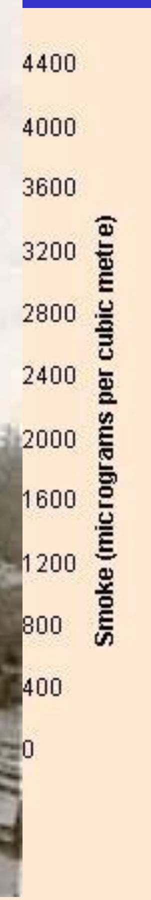
Such studies become more important as the risk due to atmospheric pollutants becomes smaller and the duration of exposure required to produce effects becomes longer

<u>Types</u>

- Cross sectional (assess effects across a cross-section of population)
- Longitudinal (assess effects over a period of time)
- Case control (compare exposed group to an unexposed, control, group)

Ultimately, it was determined that the source of London “smog” was a local coal-burning power plant

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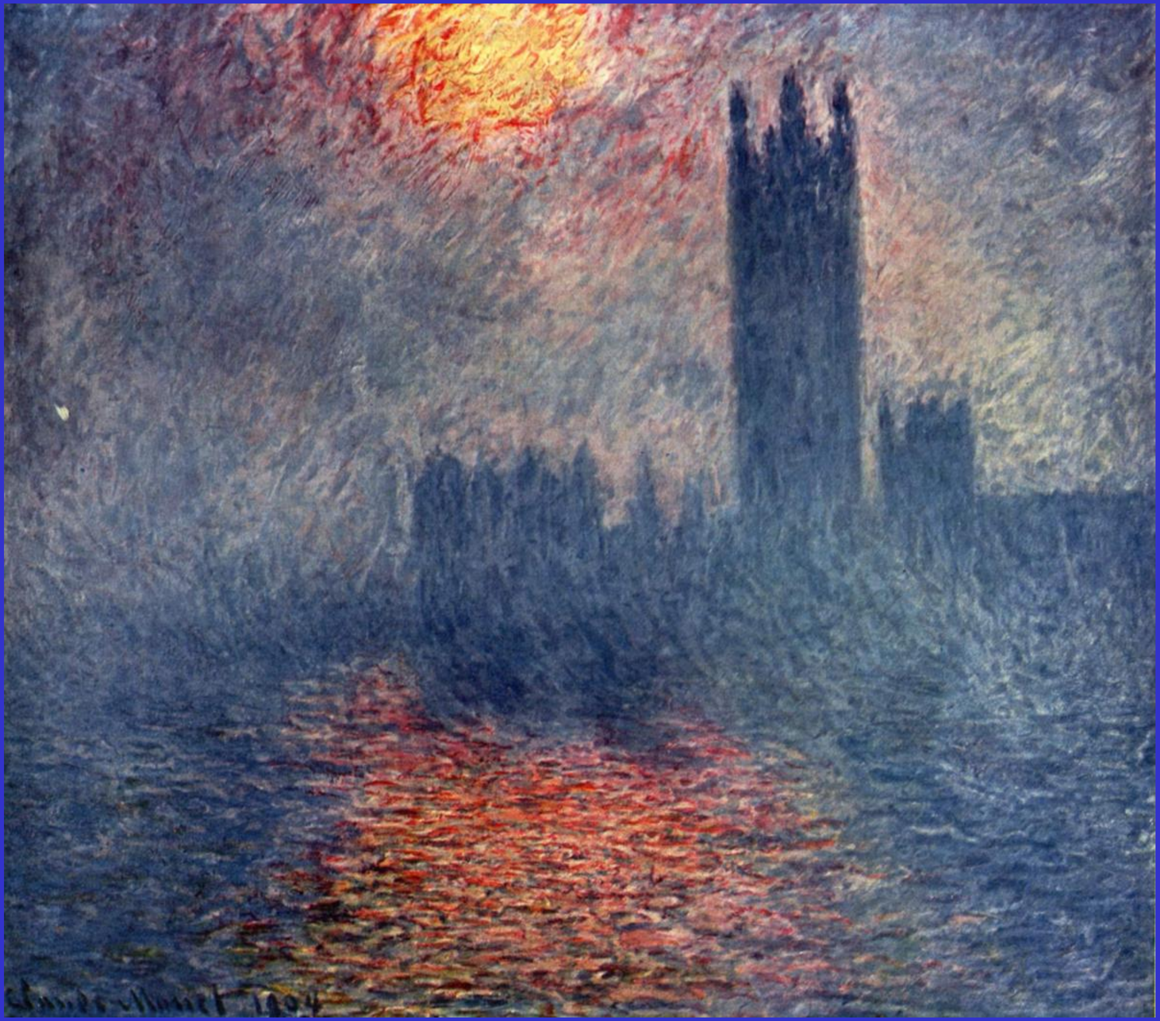
Smog was the term first used in 1905 to describe these smoke + fog events that were known to have a sulfurous smell and to cause acute respiratory illness.

Occur in areas with large primary pollutant emissions, typically from dirty burning (e.g., coal, wood, dung, etc.). Exacerbated by strong inversions, so typically worse in the cold season.

Although chemistry is involved (e.g., $SO_2 + OH \rightarrow H_2SO_4$), these are not typically photochemical events – more like acid raid, in that acids that are produced typically condense very quickly onto preexisting particles and these grow into larger 'droplets' that are highly acidic.



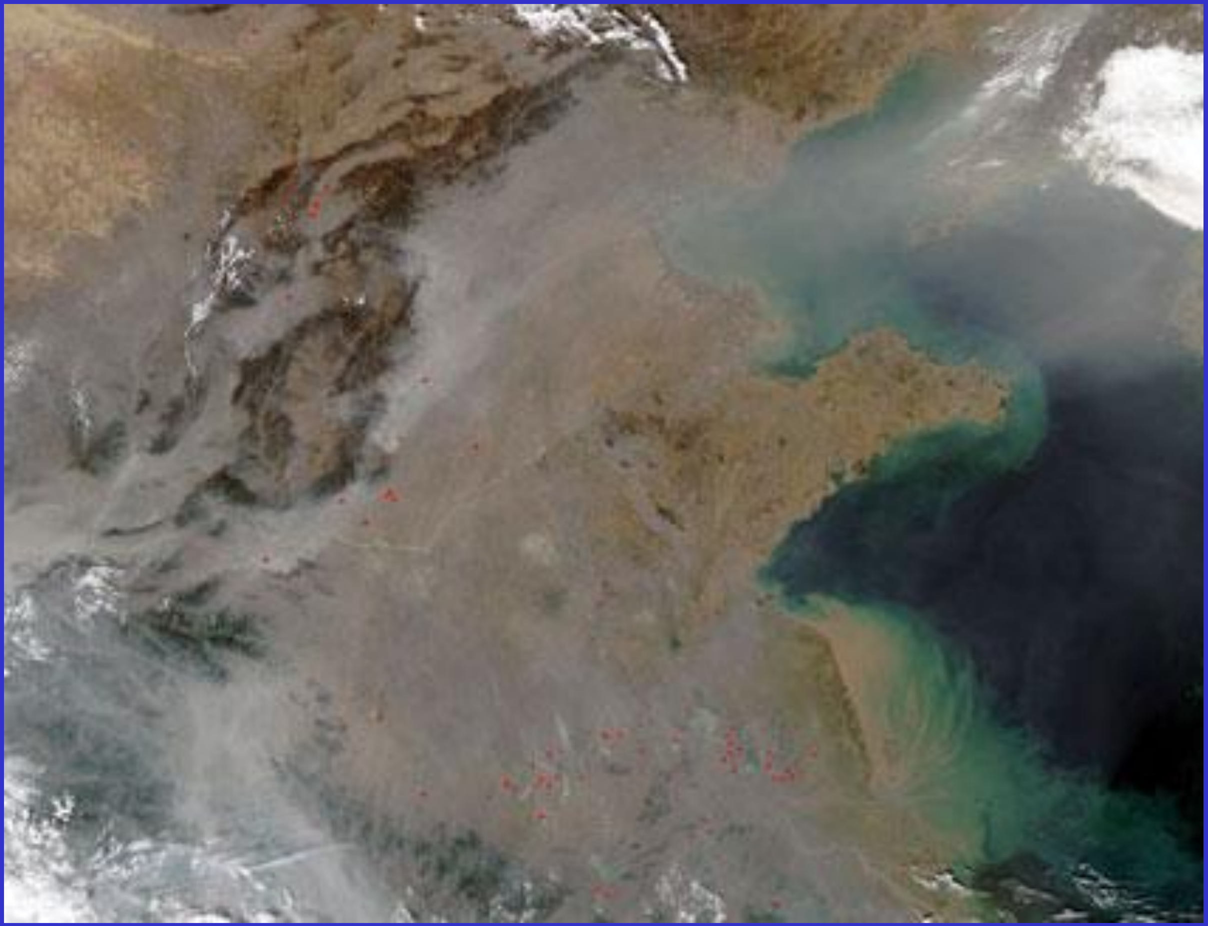
Painting of London by Claude Monet, ca 1899-1901

Kathmandu, Nepal

Mainly dust and aerosols due to vehicle traffic, HC incomplete combustion, and biomass burning

# Pollution (sulfur from coal burning) over the Great Wall of China

This pollution from China can be readily seen from space!



# Epidemiological Studies

Types

- Prospective (data to be collected)
- Retrospective (evaluation of existing data)

Confounding factors

- Multiple irritants make it difficult to isolate a single cause
- Coincidence of pollution episodes with particular meteorological conditions (e.g. temperature, humidity)
- Covariance of pollution with other exposures (eg. occupational, smoking)
- Relationships between pollution and infectious disease

# Interaction Effects

Interactions between pollutants

Example – presence of particles facilitates the respiratory uptake of $SO_2$, which dissolves in water

Effects can be additive (irritations that are similar might simply add up), antagonistic (offsetting, such as the case for ammonium and sulfate), or synergistic (new response is significantly greater than the sum of individual effects, such as with radon with smoking)

# Interaction Effects

<u>Interactions between pollutants and meteorological factors</u>

- Pollution episodes can be associated with meteorological extremes, such as cold, damp weather (London "fogs"), or heat waves

<u>Interactions between different 'types' of pollution</u>

- Smokers can be more susceptible to environmental pollution because their health is already compromised (e.g. heart disease)

- Occupational – people with different jobs may have different exercise/work habits. Americans typically spend ~2 hours per day outdoors. Someone with a job that requires them to be outdoors more frequently may experience higher levels of pollutants.

# Assessing exposure

- Personal exposure meters (often tricky to make small, but can be quite effective at isolation of pollutants)
- Exposure models
- Personal diaries

# Susceptibility

Different people are affected differently. Some are more sensitive to pollution than others:

- Elderly
- Children
- Smokers
- Those with preexisting health conditions
- Those with occupational exposure

<u>Epidemiological studies are effective when:</u>

(1) Similar effects are observed in different populations

(2) Incidence or severity of effects increases in severity with increased exposure

(3) There is a plausible biological mechanism for the effect

# Toxicity

Studies include:

- Carcinogenesis (cancer induction)
- Teratogenesis (induction of birth defects)
- Mutagenesis (production of mutations)
- Gametotoxicity (damage to sex cells)
- Endocrine disruption (hormone interference)

Usually performed on animals to minimize confounding factors

Human studies

- More definitive than those on animals
- Typically limited to acute (short term) exposure
- May not necessarily be realistic, so need to be interpreted with some caveats

ETHICS!!

# Human Health Effects

<u>Eye Irritation</u>

Exposure to aldehydes and photochemical oxidants

Although ozone and $NO_2$ don't irritate eyes, other species covary with ozone, so its abundance is used as a gauge for other species. Eye irritation typically a problem when oxidant (i.e. ozone) levels exceed ~100-150 parts per billion

<u>Cardiovascular disease</u>

Appears to be due to exposure to small particles (PM 2.5), carbon monoxide (CO) and lead (Pb)

# Respiratory Effects

Airway passageways and lungs are very susceptible to air pollution for obvious reasons

Large (> 10 μm) particles are removed in the upper airway (nasopharyngeal region) by hair and mucus. The large particles can't bend the corners of the air tract, so they impact the walls, where they are trapped and removed by cilia.

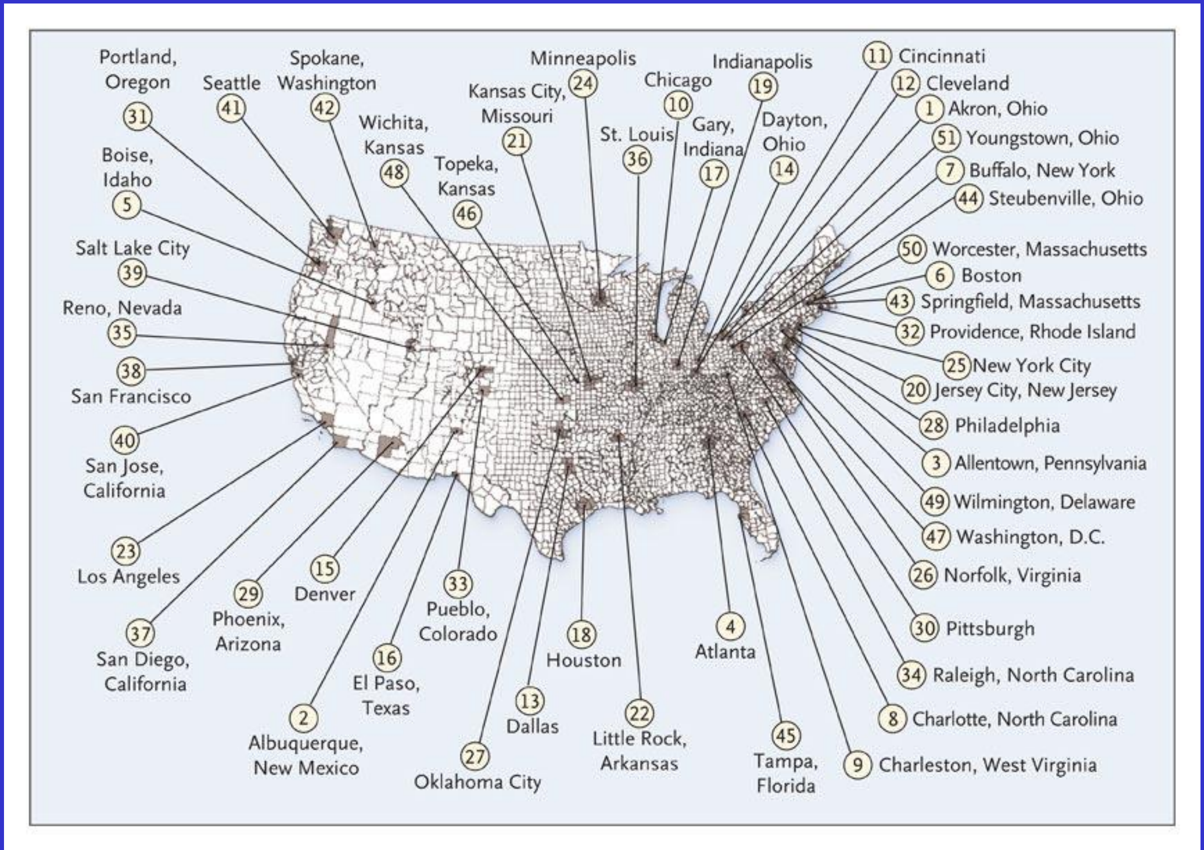
Gases ($O_2$ and $CO_2$) are exchanged in the lung tissue after traveling down the tracheobronchial region. There may be as many as 23-32 generations of branching as air travels from the trachea to the lung tissue. Cells are lined with mucus and cilia.

Gas exchange occurs in the alveoli – 300 million air sacs.

## Fine-Particulate Air Pollution and Life Expectancy in the United States

*C. Arden Pope, III, Ph.D., Majid Ezzati, Ph.D., and Douglas W. Dockery, Sc.D.*

New England Journal of Medicine, Volume 360, p 376-386, Jan. 22, 2009



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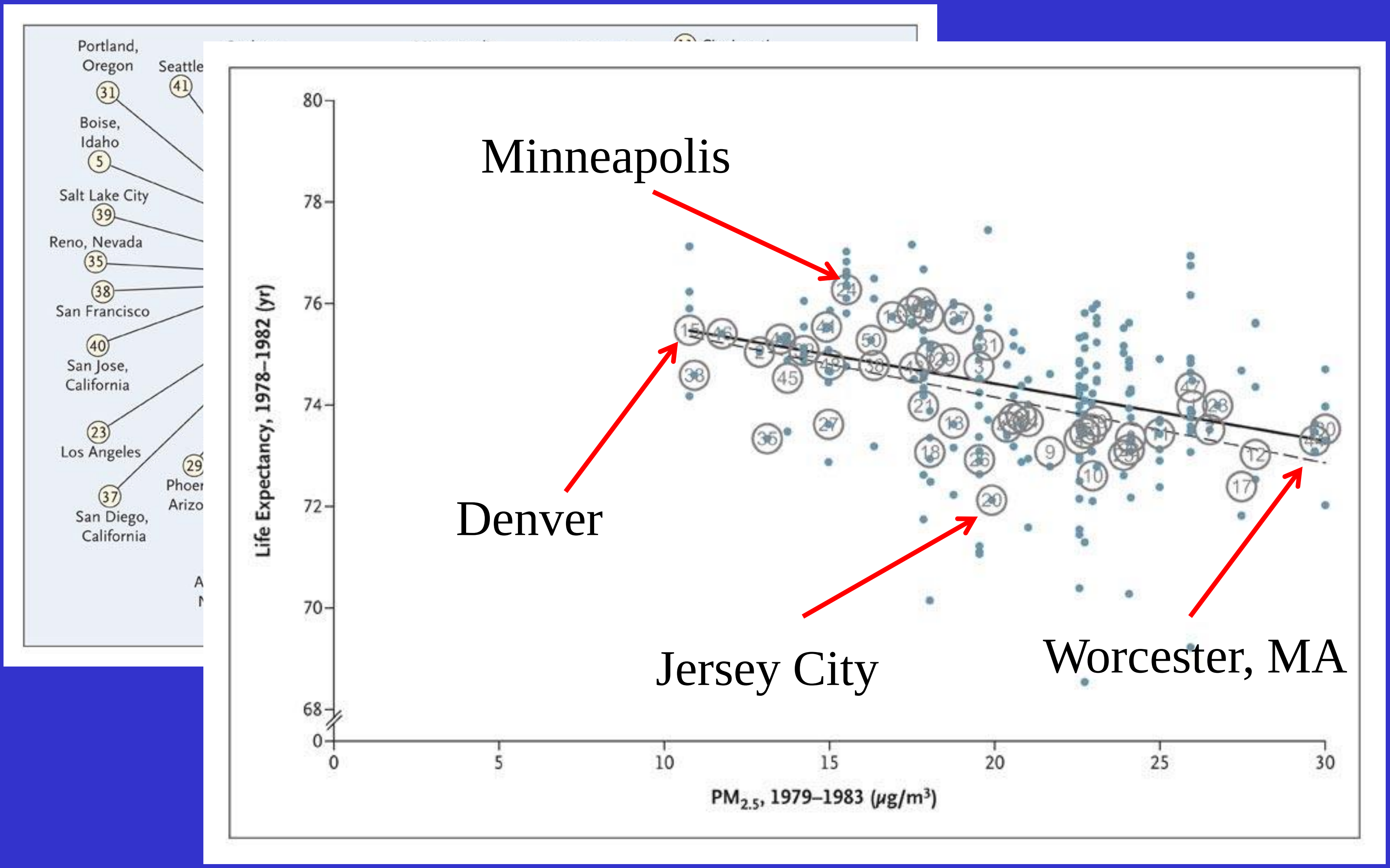

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Exposure to fine-particulate air pollution has been associated with increased morbidity and mortality, suggesting that sustained reductions in pollution exposure should result in improved life expectancy. This study directly evaluated the changes in life expectancy associated with differential changes in fine particulate air pollution that occurred in the United States during the 1980s and 1990s.

*Methods* We compiled data on life expectancy, socioeconomic status, and demographic characteristics for 211 county units in the 51 U.S. metropolitan areas with matching data on fine-particulate air pollution for the late 1970s and early 1980s and the late 1990s and early 2000s. Regression models were used to estimate the association between reductions in pollution and changes in life expectancy, with adjustment for changes in socioeconomic and demographic variables and in proxy indicators for the prevalence of cigarette smoking.

*Results* A decrease of 10 µg per cubic meter in the concentration of fine particulate matter was associated with an estimated increase in mean (±SE) life expectancy of 0.61±0.20 year ($P=0.004$). The estimated effect of reduced exposure to pollution on life expectancy was not highly sensitive to adjustment for changes in socioeconomic, demographic, or proxy variables for the prevalence of smoking or to the restriction of observations to relatively large counties. Reductions in air pollution accounted for as much as 15% of the overall increase in life expectancy in the study areas.

*Conclusions* A reduction in exposure to ambient fine-particulate air pollution contributed to significant and measurable improvements in life expectancy in the United States.

Note – study does not go lower than ~10 μg $m^{-3}$ (because there are few places that are cleaner than this…due mainly to natural sources of particulates)

Loss of about 0.6 year of life expectancy for an increase in average exposure of 10 μg $m^{-3}$ for PM2.5 (particles smaller than 2.5 μm)

Denver is a relatively clean environment, in part because it isn't that large of a city with lots of nearby open spaces. The winds also tend to blow our emissions to the east, where they become Kansas' problem.